THE PSYCHOPATHOLOGY OF APRAXIA

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It is only under special and favorable circumstances, either of experiment or of disease, that certain complex psychic or motor disturbances can be traced to an exact cerebral localization. The most interesting and at the same time the most complicated of these disturbances seem to be localized in some portion of the left hemisphere, particularly those conditions in which there seems to be a loss of the various types of speech images (aphasia) or a loss of the motor memories of the limb movements for a definite act or purpose (motor apraxia). According to recent researches, the left hemisphere seems to preponderate, in certain requested or spontaneous movements of the limbs in the same manner that it preponderates in speech. For instance in many lesions of the left hemisphere in which the right arm and leg are paralyzed, there may result a motor apraxia of the non-paralyzed left arm, thus indicating the existence of a special action of the motor centres of the left side of the brain. It seems likely that these various complex phenomena are really disorders of associative memory, either for identification or motility. There are not only different types of these disturbances but also different varieties of the same type, from the simplest to the most complex.

It is the purpose of this paper to review the recent literature on apraxia, to attempt to determine what light these studies throw upon a complicated psychic disturbance which seems to have a fairly definite cerebral localization and finally to study two cases of apraxia which have come under personal observation. The harmonizing of certain anatomical data with mental disturbances is of the greatest psychological importance, and nowhere can this be better done than in the problem of apraxia.

Apraxia was a term formerly applied to the intellectual non-recognition of objects, but the more recent investigations have shown that the term had best be limited to certain essential disorders of voluntary acts and movements. The chief disturbance in motor apraxia is an inability to make movements for the purpose demanded by the will, although the subject may understand commands and the use of objects; memory and attention may be normal and the limbs may be free from

paralysis, ataxia or tremor. Liepmann's case of unilateral apraxia which was studied clinically and anatomically in a most thorough manner, has formed the basis of the modern conceptions of the condition. In this case there was successful correlation of the clinical symptom-complex with the pathological findings. Later Liepmann extended his studies to comprise all the motor disturbances of brain disease.[1]

Previous to the work of Liepmann the ideas concerning the nature of apraxia were in a very unsatisfactory condition. It was looked upon as a form of a disorder of identification related to mind blindness. Hughlings Jackson's ideas concerning imperception or Allen Starr's description of the loss of object memories, are instances of the older interpretations. Monakow had also observed that certain aphasics showed either an inability to execute certain movements on command or misused familiar objects. Under the name of asymboly there also was described a loss of images of palpation, of kinetic movements and of the use of objects. The term asymboly has also been applied to an imperfect grasp of the nature and use of common objects, such as occurs in confusional or delirious states. In a review of the question of tactile aphasia,[2] I pointed out that the term can also be used to designate not only a failure to know the shapes of objects and their cardinal qualities, but also the ultimate recognition of the objects

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[1] The following is a bibliography of Liepmann's principal writings on apraxia.

A. Das Krankheitsbild der Apraxie (Motorischen Asymbolie) auf Grund eines Falles von einseitiger Apraxie—Monat. f. Psychiatrie u. Neurologie—Bd. VIII.

B. Der weitere Krankheitsverlauf bei dem einseitigen Apraktischen und der Gehirnbefund auf Grund von Serienschnitten—*Ibid.*—Bd. XVII and XIX.

C. Ueber Störungen des Handelns bei Gehirn-Kranken—1905.

D. Drei Aufsätze aus dem Apraxiegebiet—1908.
(This monograph is a collection of three previously published articles on apraxia—Kleine Hilfs-Mittel bei ber Untersuchung von Gehirn-Kranken—(1905). Ueber die Function des Balkens beim Handeln und die Beziehungen von Aphasie und Apraxie zur Intelligenz. (1907) Die linke Hemisphere und das Handeln (1905).

E. The section Die Krankheiten des Gehirns in Lehrbuch der Nervenkrankheiten (Herausgegeben von Hans Curschmann—1909) is by Liepmann and contains a summary of his latest views on apraxia.

F. Ein Fall von linksseitiger Agraphie und Apraxie bei rechtsseitiger Lähmung (Liepmann and Maas)—Jour. f. Psychologie u. Neurologie—Bd. X, 1908.

G. Ein neuer Fall von motorischer Aphasie mit Anatomischen Befund (Liepmann and Quensel)—Monat. f. Psychiatrie u. Neurol. Sept., 1909.

[2] The Question of Tactile Aphasia—Journal Abnormal Psychology—Vol. I, No. 6, 1907.

themselves. In its broadest sense, therefore, basing it at least upon the available data of our clinical analyses, we may say that apraxia in general is motor perplexity plus a disorder of identification. Apraxia may be divided into the motor and ideational forms. In motor apraxia the limbs do not obey the psychical wish: there is pure motor perplexity. The motor memories for movements of the limbs may be preserved but these memories are distorted, isolated or insufficiently connected with other portions of the cortex. In motor apraxia there is also a defect in the use of objects, although the objects may be perfectly recognized. The subject merely fumbles with objects; he is unable to translate a subjective purpose into an objective action. In ideational apraxia or agnosia the subject misuses objects because there is a disturbance of identification. For instance, he may think a comb is a cigar and so put it in his mouth as if smoking it. The term apraxia should therefore be limited to certain motor disorders, and it is best to refer to the ideational disturbance as agnosia.

Before the case of unilateral apraxia came under Liepmann's observation, the patient was believed to be suffering from aphasia and post-apoplectic dementia. The principal physical symptoms in this case were left facial paralysis, unequal pupils and weakness of the left leg, but no real paralysis of any of the limbs. There was pure motor aphasia and some alexia, but no word deafness, mind blindness, hemianopsia or unilateral psychic blindness. There was a marked disorder of the stereognostic sense and slight hypoesthesia of the left hand. Orientation, memory and attention were normal. The movements of the right arm were poorly executed, ill-directed and fumbling. With the left leg and arm, however, everything was correctly done. It was demonstrated, after careful study, that a typical right-sided motor apraxia was present. The analysis of this peculiar psychic condition presented many difficulties, but it was at last successfully accomplished. With the right hand responses to simple orders, such as touching the nose or buttoning the coat, and the imitation of movements were incorrectly done and with much fumbling. All commands, however, were correctly and promptly executed with the left hand. The patient blundered at every attempt to use objects with the right hand. For instance when a comb was placed in his right hand he would put it behind his ear like a penholder. Writing was defective with the right hand; the left hand produced mirror writing. For further clinical details the original monograph should be consulted.

The interpretation of the symptom complex in this case is a striking example of what may be accomplished by careful

clinical observation. Word blindness and word deafness were both absent, as all orders, movements and likewise the imitation of movements, were correctly performed with the left hand. The question may be raised that because the patient identified and correctly used objects with the left hand, and not with the right, there existed a right unilateral psychic blindness. This objection had no basis in fact, because objects placed in the right visual field were promptly identified. Because of the above condition Liepmann believed that the apraxia was not dependent on defective recognition of objects. In attempting an explanation of the condition he stated that the centripetal stimuli from the limbs on the right were perceived in the left sensory-motor area of the brain (the anterior and post-central convolutions and probably a portion of the parietal lobe), but on arriving there the impulses were blocked and therefore not transmitted to other cerebral centres. The left sensory motor area was therefore cut off from all communication with the rest of the cortex. The patient had lost the kinetic memories of movements of the right side because the left or leading hemisphere was isolated. He perceived the position, movements and tactile sensations of his limbs on the right, but was incapable of synthesizing these elements. The localizing diagnosis of the condition based on the clinical symptoms, showed how carefully the case was observed and analyzed, particularly if this be compared with the later anatomical findings. Because of the absence of word deafness, paralysis, sensory disturbances, hemianopsia, or psychic blindness, the corresponding brain centres must have been intact. The motor aphasia indicated a lesion of the third left frontal convolution and probably of the insula. The motor area and the gyrus angularis could not have been involved, because there was no paralysis and no symptoms pointing to the central optic tracts. The supra marginal gyrus and superior portions of the parietal lobe were probably destroyed. The corpus callosum was probably also involved in the process, because of the interruption of communication with the right hemisphere.

The patient died two years later from an apoplectiform attack. The anatomical findings corresponded in a remarkable manner with the earlier localizing diagnosis. The autopsy showed in the left hemisphere two foci of softening in the third left frontal convolution and a subcortical cyst in the inferior portion of the ascending parietal convolution. In the right hemisphere there was a destruction of a majority of fibres for the face and limbs on the left side, and foci of softening in the supra-marginal and angular gyri. The corpus callosum had entirely disappeared, with the exception of

the splenium. The serial sections of the brain showed these lesions with great clearness. The degeneration of the corpus callosum and of the tapetum, the softening and degeneration of the right internal capsule, the cyst of the insula and the shrunken inner nucleus of the right thalamus, were well marked. This degeneration of the corpus callosum is an important point, for Liepmann has shown that the left hemisphere is influenced by the motor region of the right cortex through the fibres of corpus callosum, and when these fibres are destroyed, the left hemisphere becomes isolated.

It was not long before Liepmann's work was confirmed by other investigators. The fundamental difference between apraxia and aphasia was recognized by Oppenheim, and later Pick reported an arterio-sclerotic and senile case in which apraxic symptoms were episodic. In a case reported by Liepmann and Maas, there was a right-sided paralysis with a left-sided agraphia and apraxia. The agraphia may be regarded as merely one of the manifestations of the apraxia. The right hemisphere was intact. In the left hemisphere, however, there was an area of softening involving the whole of the left half of the corpus callosum. In the serial sections of the brain the softening could be traced along the whole left side of the corpus callosum from the knee through the body to the splenium. In interpreting their case the authors stated that the left-sided apraxia was due either to the separation of the left hand from the memory centres of the left hemisphere, or perhaps the direct pathway for impulses to the sensory-motor area of the right brain had been destroyed. In Strohmayer's case,[1] in which aphasic and ataxic symptoms were absent, and the muscular and palpation senses intact, the patient recognized and named objects correctly, but misused them. Anatomically there was found a lesion of the left inferior parietal lobe, the supra-marginal gyrus, the forceps major and the superior longitudinal bundle. The gyrus angularis was not involved. Abraham[2] observed apraxic phenomena in two general paralytics and in a later contribution he reported a case of unilateral apraxia which on autopsy showed a lesion of the superior parietal lobe on the left. In Marcuse's case the apraxic symptoms were due to a general senile brain atrophy.[3]

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[1] *Strohmayer:* Ueber subcorticale Alexie mit Agraphie und Apraxie, Deut. Zeit. f. Nervenheilkunde, Bd. XXIV, 1903.

[2] *K. Abraham:* Ueber einige seltene Zustandsbilder bei Progressiver Paralyse—Allg. Zeit. f. Psychiatrie, Bd. LXI. 1904.

Ibid.: Beiträge Zur Kenntnis der motorischen Apraxie auf Grund eines Falles von einseitiger Apraxie—Centralbl. f. Nervenheilk. u. Psychiatrie. Mar. 1-15, 1907.

[3] *Marcuse:* Apraktische Symptome bei einem Fall von Senile Demenz. Centralbl. f. Nervenheilk. u. Psychiatrie, Dec., 1904.

Here the defects of the voluntary acts were caused by a continuous amnesia. The patient would probably forget the course of a movement after it was once started, in the same way that the tactile asymboly reported by Bourdon and Dide, was due to a kind of a continuous amnesia for tactile impressions.[1] However, very extensive amnesias may be free from any form of apraxia, as in the Lowell case of amnesia, a proof that these particular disorders of memory play little or no part in the mechanism of apraxia.[2] Pick insists strongly on the psychic element in all cases of apraxia.[3]

D'Hollander[4] has reported two cases of apraxia. The first of these occurred in a case of focal general paralysis and was bilateral. Anatomically nothing was found which could explain the condition other than the significant point that the left hemisphere of the brain was smaller than the right. In the second case, one of alcoholic dementia, there gradually developed a slight paralysis of the right arm and leg with a left-sided apraxia, thus showing the supremacy of the left hemisphere in the execution of voluntary movements. For localizing diagnosis the author suggested a lesion on the left side of the brain extending to the central semiovale, and involving the callosal fibres that go from the left to the right hemisphere.

Goldstein[5] has reported a case in which after the disappearance of a left-sided paralysis there appeared a motor-apraxia limited to the same side. For localizing diagnosis he suggested a lesion in the subcortex of the right central convolution, damaging its connections with the frontal lobe and involving the fibres of the corpus callosum. A year later, an anatomical examination disclosed, among other things, a complete destruction of the corpus callosum throughout its whole extent.[6]

In Rhein's[7] case the right hand was apraxic; apraxic phenomena were present in chewing and walking while the left hand was capable of only elementary reflex acts. The pos-

[1] *B. Bourdon* and *M. Dide:* L'Année Psychologique, 1904. (See my abstract in the *American Journal of Psychology,* April, 1905, pp. 252-254.)

[2] *Isador H. Coriat:* The Lowell case of Amnesia, Journal Abnormal Psychology, Vol. II, No. 3, 1907.

[3] *A. Pick:* Studien ueber Motorische Apraxie, 1905.

[4] *F. D'Hollander:* Bulletin de la Société Médicine Mentale de Belgique, 1907–8 (an excellent summary of all the literature, with a report of two personal observations).

[5] *K. Goldstein:* Zur Lehre von der motorischen Apraxie, Journal f. Psychologie u. Neurologie, Bd. XI. H. 4-5-6-.

[6] Der makroskopische Hirnbefund in meinem Falle von linksseitiger motorischen Apraxie, Neurol. Centralblatt., Sept. 1, 1909.

[7] *J. H. Rhein:* A case of apraxia with autopsy. Journal of Nervous and Mental Disease, Vol. 35, Oct., 1908.

terior portion of the corpus callosum was found degenerated at autopsy.

In Vleuten's[1] case, there was a sarcoma in the left hemisphere, which by invasion and pressure destroyed the cingulum, the whole of the left half of the corpus callosum and part of the right genu. The right hand and arm was tremulous, while the left hand and arm showed apraxia. Here the left-sided apraxia was produced by a lesion which destroyed the callosal fibres. Bychowski[2] has reported a case in which the apraxia was found to be due to a cyst in the left hemisphere which destroyed and displaced a portion of the left side of the corpus callosum. In Hartmann's[3] three cases of apraxia, one was due to a tumor in the left frontal region, and the second to a tumor involvement of the corpus callosum. In a case recently reported by Tooth[4] there was an inconstant motor-apraxia of the left hand. At autopsy there was found a tumor occupying a portion of the right frontal lobe, involving the anterior half of the corpus callosum.

In discussing the psycho motor disturbances of various mental diseases Kleist has shown how apraxic phenomena may appear in the hyperkinetic and akinetic motility psychoses. He thinks that these disturbances are probably due to psychic factors and that they may have a definite cerebral localization, particularly in the parietal lobe.[5] This is of interest if we remember that in some cases of unilateral motor apraxia, the parietal lobe was found involved as well as the corpus callosum. For instance in one case of an akinetic motility psychosis which came under personal observation, it was noted that the subject had lost all knowledge of the use of simple objects. Stransky[6] has shown how apraxic phenomena may appear in dementia præcox. In these cases he interprets it as due to a loss of unity between the understanding and the will.

Apraxia may also occur as a disorder of consciousness in delirium and post-epileptic states and under both these conditions without any definite focal lesion. Here the apraxia

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[1] *C. F. V. Vleuten:* Linksseitige motorische Apraxie, Ein Beitrag zur Physiologie des Balkens, Allg. Zeit. f. Psychiatrie, 1907.

[2] *Z. Bychowski:* Beiträge zur Nosographie der Apraxie—Monat. f. Psychiatrie u. Neurologie. Bd. XXV, 1909.

[3] *F. Hartmann:* Beiträge zur Apraxielehre, Monat. f. Psychiatrie u. Neurologie. Bd. XXI, 1907.

[4] *H. H. Tooth:* (Abstract in Review Neurology and Psychiatry, July, 1909, pp. 475-476.)

[5] *K. Kleist:* Untersuchungen zur Kenntnis der psychomotorischen Bewegungsstörungen bei Geisteskranken, 1908.

[6] *E. Stransky:* Zur Auffassung gewisser Symptome der Dementia Præcox, Neurol. Centralbl., Dec., 1904.

is ideational, in the sense of a disorder of indentification. In these cases the phenomena tend to disappear as the mental state improves. In my study of a delirious state associated with vestibular disturbances, apraxic phenomena were present.[1]

Motor apraxia has been found to occur in aphasia. The disorders of movement which one frequently finds in aphasics are very likely not due to any intellectual defect as claimed by Marie, but to a disorder independent of aphasia, namely apraxia. Cases of dementia very rarely show symptoms of apraxia. It cannot be said that apraxia is due to any intellectual disorder because in disturbances of motility where the apraxia is limited to one side of the body, the other side of the body will be found to be absolutely normal. This shows that intellectual defects may be practically ruled out in motor apraxia, at least in the unilateral types, unless it is absurdly assumed that only one side of the brain is demented. In one of my cases of sensory aphasia the patient was bright and alert and yet he ridiculously insisted on eating an egg, shell and all. Here we have an example of apraxia occurring in an aphasic subject without mental defect. Ideational apraxia (agnosia), however, may occur in certain abnormal mental states, such as in delirious conditions or in multiple brain lesions.

There are many striking points of similarity between motor aphasia and motor apraxia. The motor speech mechanism is really a form of movement without objects. The centre for motor speech is located in the left hemishpere and it has also been shown how the kinetic memories for co-ordinated movements likewise preponderate in the left hemisphere, at least in right-handed subjects. In complicated movements which through habit and evolution, have become bilateral, such as the lip-tongue-larynx movements in speech, there is usually a loss of these movements in a lesion of the motor speech area. In the limb movements which as a rule are not bilateral, but in which right and left are independent and in marked contrast, there results a unilateral apraxia when a lesion on one side of the brain is favorably situated.

How then are we to explain these complex phenomena and what light can be thrown upon them by brain anatomy and physiology? Certainly the subject of apraxia opens up one of the most inviting and at the same time one of the most difficult fields in psychopathology. At first it will be well to attempt to analyze apraxia on the basis of the data furnished by those cases where it was possible to make an anatomical examination of the brain.

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[1]The Cerebellar-Vestibular Syndrome, American Journal of Insanity, Vol. LXIII, No. 3, Jan., 1907.

The point of significance in the majority of apraxic cases was the involvement of some portion of the corpus callosum. The corpus callosum must then be possessed of a definite function, as its involvement seems to have been as invariably present in motor apraxia as a lesion of Broca's convolution in motor aphasia or of the central optic tracts or the cuneus in hemianopsia. What then is the function of the corpus callosum and how are we to harmonize the motor and psychic disturbances with the anatomical findings?

The fibres of the corpus callosum connect the two opposite sides of the brain. In fact in experimental lesions of the thumb centre in monkeys, the degenerated fibres could be traced directly through the corpus callosum to a similar area on the hemisphere of the opposite side. The knee of the corpus callosum sends its fibres to the fore brain as the forceps anterior. The fibres of the splenium go to the hind brain (occiptal region) and to the temporal lobes as the forceps posterior and make up the greater part of the tapetum. The body of the corpus callosum connects the two hemispheres in the mid position. Both the left and the right hand centres, corresponding respectively to the movements of the right and left hands, are connected by means of the fibres of the corpus callosum.

By reason of its wide connections, the corpus callosum plays an important part in the execution and association of voluntary movements. In tumors of the corpus callosum, disorders of movements are frequently seen, such as apraxia, tremor and ataxia. In a case of tumor of the splenium of the corpus callosum which came under personal observation, there was a paralysis of the right arm and of the right side of the face and a continual coarse tremor of the left arm. A lesion of the corpus callosum alone causes a left-sided motor apraxia without paralysis or apraxia on the right. A lesion of the left hand centre causes paralysis on the right side and apraxia on the left. It seems that certain left-sided lesions produce a left-sided apraxia because the control of the right hemisphere over the innervation of the left arm by means of the corpus callosum has been cut off. The left hemisphere thus becomes isolated and leaderless.

The following diagram will indicate the various brain lesions which may produce motor apraxia.

(See figure I.)

A lesion at I (the left brain centre for the right arm) will produce a paralysis of the right arm and an apraxia of the left arm because this centre is either isolated or is deprived of the guidance of the right hemisphere. A lesion at II (in the subcortex of the left Rolandic area), injuring the

projection and callosal fibres to the right hemisphere, will also produce a paralysis of the right arm and an apraxia of the left for the same reason as indicated under lesion I. A lesion at III (the left internal capsule) will cause a right-sided paralysis, without any apraxia on the left, because the corpus callosum is not injured. A lesion at IV (in the corpus callosum) will cause a left-sided apraxia. A lesion at V (in the left centrum ovale, catching only callosal fibres) will likewise cause a left-sided apraxia. In both these conditions,

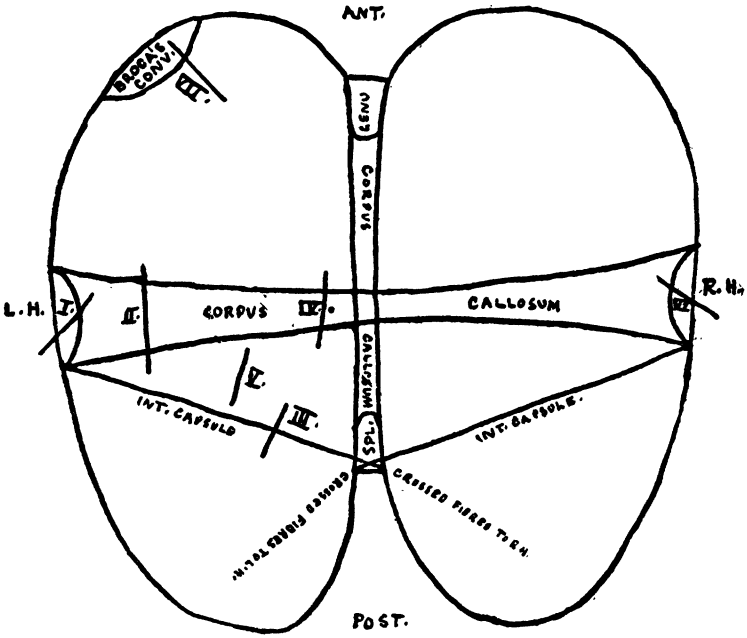

Fig. I

A diagrammatic horizontal section of the brain, to illustrate the anatomical basis of motor apraxia.

L. H. Left brain centre for right hand.
R. H. Right brain centre for left hand.

The anterior and posterior and the lateral course of the fibres of the corpus callosum are shown diagrammatically.

the apraxia is due either to a loss of the guiding influence of the right-hand centre over the left hand or to an isolation of the hand centre from the rest of the left hemisphere. A lesion at VI may cause a right-sided apraxia from an interruption of the callosal fibres passing to the left side of the brain. (Liepmann's first case.) A lesion at VII (Broca's convolution) would

produce motor aphasia, and if the lesion be of sufficient size and extent to catch the radiations of the genu of the corpus callosum (or forceps anterior), there will be likewise caused a left-sided motor apraxia. That this latter combination is not impossible, is shown by the case recently published by Liepmann and Quensel and also by the second of my reported cases.

We must admit that the psychical condition of apraxic subjects presents great difficulties of analysis, even more so than the analysis of disturbances of the language mechanism in the various types of aphasia. Probably with increasing knowledge of apraxia some of these difficulties may be overcome. Motor apraxia in its strictest sense is rarely bilateral as can be easily seen from the reported observations. It is in ideational apraxia or agnosia that the bilateral nature of the disturbance may be detected. In fact, agnosia seldom or never occurs as a pure isolated disorder in focal brain disease. When it does occur in focal disturbances of the brain, it is merely as a complication of the motor type of apraxia. Agnosia is most frequently found in diffuse brain affections (senile dementia), in delirious states particularly due to epilepsy or alcohol or in the motility psychoses. Hence agnosia is a more general disturbance, while apraxia is a focal disorder limited to certain limbs or to muscle groups.

In motor apraxia the limbs do not obey the psychical wish, although that wish and the motor image of the willed movement may be clearly present in the mind of the subject. It is this inability, on the part of the subject, to transfer the psychical wish into a specified innervation of a limb, which causes motor apraxia. In ataxia it is the elementary condition of the movements which is at fault, while in apraxia there exists a disharmony between the purpose of the movement and the idea of the object with which the purpose is carried out. In other words, in motor apraxia there is a deficient adjustment to a purpose. In motor apraxia the coordination of movements is normal, but there is a faulty intrapsychic process and so the ultimate purpose of these movements becomes incorrect. The motor apraxic cannot translate the subjective idea of a movement or of the use of an object into its objective reaction. Motor apraxia therefore may originate from either an insufficiency of the directing idea, from derailment or irradiation of the directing idea upon the neighboring ideas, from the omission of portions of the motor act and the predominance of the last portion of the act or from the substitution of the directing idea by another idea.

All normal voluntary activities seem to be due to two

mechanisms—the representation in the sensory sphere of the successive series of partial acts which make up the entire act (the kinetic formula for movements) and the faculty of changing this sensory representation into external movements (the ability of exteriorization). The kinetic formulæ for movements are really the memories of successive movements and can be compared to certain chain or sequence reflexes, a point upon which I had previously insisted in my studies on amnesia. In motor apraxia, this kinetic formula is defective. Therefore it acts in an abnormal manner upon the innervation of the specific action, according to which of the kinetic elements is disturbed. Motor apraxia consists, therefore, in a rupture of the physiological connections between the innervation of a specified limb and the ideas concerned in the carrying out of a specified purpose. It is really a motor dissociation, in which special kinaesthetic memories lie outside of the field of general motor innervation. This isolation of the kinæsthetic memories is probably due to an isolation of a certain portion of the left hemisphere in which these motor memories are stored up, an isolation resulting in most cases, from a rupture in the conduction of the fibres of the corpus callosum. The centripetal stimuli reach the centrifugal pathway without entering the special ideational or motor centres by a kind of "short circuit." This is pure motor apraxia; when in addition there exists a disorder or identification, we have motor apraxia plus asymboly or ideational apraxia (agnosia). In all cases of motor apraxia the chief difficulty seems to lie in the inability of the subject to transfer the directing ideas or rather the willing of the directing ideas to the motor sphere because they are partially cut off, isolated or derailed.

The behavior of apraxic patients is a subject of much interest. Movements of substitution which are so common in motor apraxia, may be compared to paraphasia. Occasionally the subject may begin a movement and only partly complete it. Here the reaction is referred to as curtailed. Sometimes a movement may bear no resemblance whatsoever either to the usual acts of everyday life or to a specially skilled reaction. Unde these conditions we describe it as a formless or an amphorous reaction. On still other occasions the first part of the movement may be immediately followed by the last part without any intervening motions. This is called a short circuited reaction ("Kurzschluss Reaktion"). Perseveration is the monotonous automatic repetition of an act. Sometimes indeed the subject becomes petrified, as it were, in the attitude of executing a simple or a complex act either requested or spontaneous. To the first form is applied the term clonic perseveration; the latter is known

as tonic perseveration. Clonic perseveration strongly resembles the recurrent utterances of sensory aphasia, while tonic perseveration is analogous to the mutism of a severe motor aphasia.

Scheme of disturbances of kinetic formula for movement.

A —⟩ B —⟩ C —⟩ D = Normal Motor Reaction.
A ————————⟩ D = Short Circuited Reaction.
A —⟩ B = Curtailed Reaction.
A —⟩ A —⟩ A—⟩ = Clonic Perseveration.
A = Tonic Perseveration.

FIG. II.

A graphic analysis will make this clearer (see figure II). Let A. B. C. D. represent the kinetic formula for a certain movement, each letter indicating one element of the action. The normal movement could occur only when the individual elements acted serially. Any change in the position of the elements or any omission, would result in disordered activity or apraxia. For instance if the action took place as A-D. with the intervening elements omitted, we would have a short circuited reaction. If the movement was A—B. with the other two elements omitted, then the action ceases soon after it has been started; in other words, it has become curtailed. If the action should be A. A. A, the first portion of the action will then tend to be indefinitely repeated. A clonic perseveration could result. If the action was A. alone, and no further elements of the kinetic formula followed, the subject then would become stuck at the first of the series. Here we have a tonic perseveration. Many apraxic subjects show a marked lack of spontaneity, a condition which strongly resembles, if it is not identical with tonic perseveration. As a rule most requested or spontaneous movements in apraxic subjects are ill directed and fumbling, show a deficient adjustment to a purpose and yet they are not ataxic.

Von Monakow[1] has brought forth an ingenious theory to explain certain focal disturbances of the brain, particularly aphasia and apraxia. According to this theory, mere anatomical interruption of the continuity of fibres in the central nervous system will not fully explain the various form of focal disorders. Therefore there must be a special form of action at a distance from which may arise temporary or permanent suspensions of function. To this action at a distance, von Monakow applies the term "diaschisis." This diaschisis resembles certain physiological irradiations, in which a reflex effect can spread in various directions from a focus of reflex discharge.

[1] *Von Monakow:* Neurol. Centrablatt, Nov. 16, 1906.

The study and analysis of two cases of motor apraxia which came under personal observation will now be taken up. The difficulty of correlating the motor reactions with the mental state of the subject will excuse the lengthy details of the reports. In fact, in apraxia as well as in aphasia, a rigid examination scheme has but a limited value, as not only do the types and the conditions vary, but the same type may present different aspects in different subjects. Our present knowledge of apraxia is due entirely to minute clinical investigations, to which, when possible, the anatomical findings have been added.

Case I. When the patient K., who was twenty-four years of age, first came under observation he had been suffering from severe headaches for two months. On several occasions the headaches became so intense that vomiting followed. Up to this period the patient had been perfectly healthy. An examination disclosed the following condition. Only the essential neurological details are given. Tongue tremulous and protruded to the right, tremor of outstretched hands, the right knee jerk and right Achilles jerk were brisk; while the left knee jerk and the left Achilles jerk were absent. There was a subjective sense of weakness and numbness on the left side of the body. There was no ankle clonus or Babinski reflex. The pupils were equal and reacted promptly to light and accommodation; there was no paralysis of the ocular muscles and no nystagmus, but an ophthalmoscopic examination disclosed a blurring of the optic disc without swelling (beginning optic neuritis).

About three weeks later a sudden but transitory paralysis of the left arm and leg took place. Within a few days this improved until only a slight weakness could be detected. The physical condition has since remained the same with the exception of a blunted sensation in the left hand, associated with a weakness of grasp. Word deafness and motor aphasia were absent during the entire course of the disease. The patient was right-handed.

There was no hemianopsia and no unilateral mind blindness, because objects placed in each visual fields were promptly recognized. An analysis showed a typical motor apraxia of the left arm, while the right arm was entirely free from any motility disturbance. This unilateral apraxia was not dependent on any defective recognition of objects, because the patient could correctly select objects with either hand, but did not know how to correctly use them with the left hand after they had been selected. He was always oriented, and there was no disorder of memory either for recent or remote events. Unilateral agraphia was absent; he was able to write spontaneously and to dictation fairly well with either hand.

There was no apraxia of the left leg or of the muscles of facial expression for either side. This appearance of a motor apraxia in a limb from which a previous transitory paralysis had disappeared, resembles the observation of Goldstein, to which we referred above.

Examination for Apraxia, Motility and Sensation of the Left Arm

The general movements of the left arm were weak and fumbling. The dynamometer for the right hand registered seventy, while for the left hand it was twenty. The left arm was not paralyzed and all the movements could be fairly well performed but they were ill-directed and awkward. The abnormal motor reactions of the left arm on analysis seemed to be entirely due to a motor apraxia and not to any motor weakness or ataxia. With the eyes closed the movements of the left arm were decidedly more fumbling and ill-directed than when the eyes were open. In spite of the fumbling and slight weakness and spasticity of the left arm, he could spontaneously lift it above his head, flex, extend, pronate, supinate, fairly well extend and flex the fingers and give a fair grasp, although there was a disinclination on the part of the subject to spontaneously use this left arm. This tendency to akinesis was probably due partly to the patient's appreciation of his localized motor apraxia and partly to a state of tonic perseveration. An examination of the sensation of the left arm, forearm and hand showed anæsthesia and hypoalgesia.

The testing of the muscular sense of position showed that when the right forearm was placed at a right angle to the right arm, after some fumbling he was able to correctly imitate the position with the left arm. When the right arm was placed vertically above the head he promptly placed the left arm in the same position. With the eyes closed, when the right arm was passively elevated above the head and the patient was requested to imitate the position with the left arm, he merely placed the latter horizontally on the same level with the shoulder. Right arm passively extended horizontally forward with the palm upwards; he imitated it by placing the left arm in the same position but with the palm downwards. Right index finger placed at right angles; with the left hand he merely made a fist.

Reaction to Requests

	R. Hand	*L. Hand*
Buttoning coat	Correct	Grasps edge of coat
Touching tip of nose with right forefinger	"	Puts palm to mouth
Touching right ear with right fore finger	"	Places finger back of head

	R. Hand.	*L. Hand.*
Movement of sewing	Correct	Fumbles
Movement of cutting with scissors	"	Fumbles
Snapping fingers	"	Makes fist
Turning hand-organ	"	Makes fist
Movement of turning key in lock	"	Makes fist
Movement of shaving	"	Places hand behind ear
Movement of combing hair	"	Rubs head with palm of hand
Movement of use of cork-screw	"	Fumbles
Military Salute	"	Fumbles

In the three successive reactions with the left hand in which the patient made a fist in response to different requests, we have an example of clonic perseveration or the frequent monotonous repetition of an act.

Reaction to the Use of Objects

	R. Hand	*L. Hand*
Comb	Correct	Rubs hair with smooth back of comb
Tooth brush	"	Correct but fumbles
Hair brush	"	Holds back of brush several inches above head without any brushing motion
Key	"	Cutting movements as though it were a knife
Match	"	Correct but fumbles
Spoon	"	Cutting and stabbing movements
Cigarette	"	Grasps it clumsily, puts it to chin and then puts wrong end in mouth

In the reaction to requests and to the use of objects the left-sided apraxia became more marked when the patient's eyes were closed. The same fact was noted in testing for the sense of position in the apraxic limb. For instance, in one series of tests a key, a match and a drinking glass were used correctly with the right hand and decidedly awkwardly and fumblingly with the left. When attempts were made to use these same objects with the eyes closed, the left hand seemed to become petrified, as it were, after an abortive start. In other words we seem to have here the phenomenon of tonic perseveration. On other occasions, even with the eyes open, he would start to use an object correctly with the left hand, and then would hopelessly fumble, either going into a condition of tonic perseveration or would finish up with a substituted movement, as if the object or the request had been changed. Sometimes, too, in the use of objects with the left hand, the movements would bear no relation to the nature of the object; they would become decidedly amorphous. The stereognostic sense was entirely lost on the left hand and to a certain extent, at least for larger and coarser objects, on the sole of the left foot. The increase of apraxia when the eyes were closed, was not due to any astereognosis, because the patient was always told the nature of the object. At no time

was there any loss of the knowledge of the use of objects. He was always able to describe their uses and from a number of objects spread before him he was able, on request, to pick out the correct one with either hand. The patient always knew when he used an object incorrectly with the left hand. The apraxia seemed therefore not dependent on any defective recognition of objects; in other words, it was not ideational but was almost entirely a motor disorder. His knowledge of the use of objects was always correct, even when he was not permitted to touch the objects.

In order to further show that there was no disorder of identification and that the inability to correctly use objects with the left hand was purely a motor disturbance, the following observations are of interest. When the patient was shown a match and requested to describe its use, he replied "To make fire." When asked to show the use of a match he did so correctly with the right hand. With the left hand, however, he clumsily grasped the entire match in his fist, leaving only a small portion of the head exposed and then made stabbing instead of scratching motions with it. When a cigarette was given to him, with the right hand he correctly placed it in his mouth. With the left hand he grasped the cigarette so clumsily in his fist that it was almost broken in half, then he placed it to his chin, then after some hesitation brought his fist to his mouth, still holding the cigarette tightly and with the mouth end completely covered up with his hand. When a match-box was placed in the left hand he promptly opened the cover, took out a match and showed correctly how to use it. When the match-box was placed in the right hand, attempts to repeat the performance with the left hand brought out a very typical motor apraxia. After considerable fumbling and perplexity he opened the cover, took out each match separately and allowed each to fall back into the box without any attempt at scratching them. When a pipe was placed in the right hand he promptly put the stem in his mouth. With the left hand, however, he took it by the bowl and placing the bowl to his chest said "I know it is a pipe, but I can't use it with this hand." It appears that these details point out the significant fact that the inability to use objects with the left hand was due not to any lack of knowledge or identification of the object, but to a derailing of a motor wish into a false motor reaction.

The imitation of movements also brought out a left-sided motor apraxia as follows:

	R. Hand	*L. Hand*
Saluting	Correct	Fumbles
Shaving	"	Merely places hand on cheek
Shining shoes	"	Grabs ankle

When requested to perform these same actions with the left arm and the eyes closed, fumbling and lack of direction in movements became greatly increased. It seems from these observations that visual impressions could partially correct the left-sided motor apraxia. When the visual stimuli were cut off by having the patient close his eyes, he went completely off the rails. The movements of the left hand without objects, were as apraxic as when objects were used.

In this case we are probably dealing with a brain tumor for with the following localizing diagnosis may be suggested. The tumor is probably in the right motor sub-cortex or centrum ovale, involving a portion of the fibres of the pyramidal tract and of the parietal region and probably a large portion of the fibres of the corpus callosum. A lesion here from its position could cause the motor disturbance of the left side of the body, that is, a transitory paralysis and weakness of the left arm and to a slighter extent of the left leg, and also a typical motor apraxia of the left arm. This latter is due to a loss of the guiding and directing influence of the right arm centre upon the left arm through a destruction of the fibres of the corpus callosum.

In the second observation we have the combination of an aphasic speech disturbance and a left-sided motor apraxia, both occurring in a right-handed subject. Such a combination is of importance, for it demonstrates that a left-sided brain lesion may be so situated as to cause an aphasia and a left-sided apraxia.

Case II. The patient L., 57 years of age, a right-handed man, began to suffer with a severe headache localized on the left side of the head, combined with dizziness and a weakness of the right arm. Shortly afterwards he suddenly began to repeat "oilcloth, oilcloth" spontaneously and in reply to all questions. This recurrent utterance lasted about an hour, after which speech became normal, although a little dysarthric. Somewhat later that same day he again suddenly began to talk in a jargon. In this jargon German sounds (his native language) predominated, while all knowledge of English (an acquired language) was completely lost. This condition lasted for four days and during this time the left-sided headache, dizziness, weakness and numbness of the right arm and leg continued although no actual paralysis was ever noticed. All requests and commands were understood. He knew the hours for meals and his various wants of his everyday life. At the end of four days he suddenly regained his normal speech in both languages. Since then when fatigued or frequently in the late afternoon he will forget the names of objects in English and will call them only by their German names. This is a

feature of interest which I had previously pointed out in the Lowell case of amnesia. It is indicated that the acquired memories in certain forms of amnesia were the first to disappear, while the deeper and more closely knit associations were preserved.

The neurological examination may be briefly summarized as follows: Marked arterio-sclerosis with a high blood pressure, speech occasionally dysarthric, no hemianopsia, grip of right hand decidedly weaker than that of left, no paralysis, pupils slightly unequal, the right reacting slightly to light, the left rigid, the right knee jerk diminished as compared to the left, no Babinski reflex, no facial paresis or deviation of the tongue. The gait was weak but not hemiplegic while the station was normal. There were no sensory disturbances in any of the extremities. All objects were quickly and correctly recognized and named and their uses accurately described. The amnesic aphasia was episodic and usually appeared only after the fatigue of a long examination or in the late afternoon. The patient was clearly oriented and showed no signs of intellectual defect.

An examination of the motility of the left hand showed that while it was slightly weaker than the right, it was free from paralysis, ataxia or tremor. All voluntary movements were present, yet it was possible to demonstrate a typical left-sided motor apraxia. The false motor reactions of the left hand were due to a motor apraxia and not to any inability to understand requests, because there was no intellectual defect and no word deafness. The facial muscles were free from apraxia. The inability to name objects, although he could indicate their uses with the right hand, took place only during the temporary anmesic aphasia due to fatigue. Repetition of printed letters was normal. Copying was correctly done with each hand. All objects were correctly recognized in each visual field and therefore there was no unilateral mind-blindness. Astreognosis was absent. The apraxia may be tabulated as follows:

REACTION TO REQUESTS

	R. Hand	*L. Hand*
Making a fist	Correct	Correct
Spreading the fingers	"	Shows palm of hand
Using cork-screw	"	Fumbles, first part of action correct, then makes cutting instead of pulling movements
Saluting	"	Correct
Smoking a cigar	"	Saluting movements

The attempt to show the use of a cork-screw with the left hand is an example of a curtailed reaction, while the persist-

ence of saluting movements to different requests indicates a clonic perseveration.

REACTION TO USE OF OBJECTS

	R. Hand	*L. Hand*
Scissors	Correct	Fumbles but correct
Key	"	Turns it upside down and holds it there
Match	"	Holds it like a pencil and makes writing motions
Pencil	"	Correct
Shoehorn	"	Rubs it against leg

A number of other objects were correctly used in the left hand but in a fumbling and awkward manner. The imitation of movements was also somewhat apraxic on the left. The imitations of movements of the right arm with the left arm led to some interesting reactions. When the patient was requested to make certain movements with the left arm alone, an apraxia always resulted, but this apraxia practically disappeared if the patient was allowed to imitate the movements of the right arm with his left. The apraxia to imaginary movements or in the use of objects was increased when the eyes were closed; or if apraxia was absent when the eyes were open, it tended to appear when the same tests were made with the eyes closed. These observations, demonstrated as in the previous case, that visual impressions are able to partially correct a motor apraxia in the same manner that a subject is less ataxic when the eyes are open.

For an anatomical localizing diagnosis we would suggest a probable area of softening at the angle of the third left frontal convolution and the Sylvian Fissure, extending below to the white matter of the corona radiata and to the radiations of the genu of the corpus callosum (forceps anterior). A lesion here would cause a motor aphasia and also a motor apraxia of the left arm, because the guiding influence of the right side of the brain upon the left side of the body would be cut off. The motor centre would thus become isolated. Here again we see the importance of the integrity of the callosal fibres from preventing any motility disturbance. The weakness and numbness of the right arm is probably due to either a backward extension of the lesion or to a pathological irradiation involving the anterior central convolution on the left.

Any analysis of these two cases demonstrated that the chief difficulty lay in an inability to transfer a subjective choice process into an objective reaction. The cause of this disorder could be easily traced to a definitely localized lesion in the brain, which disturbed the kinetic memories for move-

ments and produced new and abnormal combinations. The disordered movements and misuse of objects could be partially corrected through visual impressions, probably because these impressions may have stimulated certain non-affected portions of the brain, to function in a normal manner.